and the pleura the abdominal breathing is one-sided, whereas the influence on abdominal movement in pericarditis is bilateral, and hence absence of abdominal movement in pericarditis is as obvious as in abdominal conditions, though rigidity and tenderness are for the most part lacking in pericarditis.

He reports two cases in which remarkable stillness of the abdomen led to the diagnosis of perforated gastric ulcer in one case and of appendicitis in the other. The case of supposed appendicitis was operated upon and a normal appendix was removed without adhesions.

He concludes that in a case in which the loss of abdominal movement is marked in the absence of an obvious lung or abdominal lesion, pericarditis should be considered.

The explanation given by Mackenzie[5] for the transference of pain in visceral diseases is as follows:

1. Stimuli which produce pain in the external body wall are not adequate to produce this sensation when applied to the viscera

2. Violent contraction of a non-striped muscle fiber produces pain, but that the region in which the pain is felt is different from that in which the contracting muscle lies, and that when a sensory muscle stimulated in any part of its course in the brain, the cord, or the trunk of the nerve the pain is distributed to the peripheral distribution of the nerve in the external body wall. This is his so-called viscerosensory reflex.

He further considers that in order to call into effect this viscerosensory reflex there must be an adequate stimulus, and one may consider that because the stimulus is rarely adequate in pericarditis, pain in this condition is not always transferred either to the arm, the epigastrium (which is the most common seat of referred pain) or to the lower abdominal wall, which is the rarest seat of localization of the pain.

His visceromotor reflex may explain the rigidity of the abdominal wall in these cases.

PERICARDITIS IN CHRONIC NEPHRITIS.*

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Although the occurrence of acute pericarditis in advanced renal disease has long been known the mechanism of its origin is still obscure. Interest in the subject was aroused by a recent case. The patient was a girl, aged nineteen years, suffering from an end-stage chronic nephritis. After the development of a severe acidosis

[5] Diseases of the Heart.

* From the Medical Clinic of the Presbyterian Hospital, New York.

and a fibrinous pericarditis she finally succumbed. When at the suggestion of Dr. Longcope other cases of acute pericarditis in nephritis were reviewed it was found they fell into a recognizable group having fairly well-defined clinical and laboratory characteristics. The pathogenesis of the pericarditis was further studied by correlating the bacteriological with the pathological findings. The facts brought out were thought to throw an additional light upon the etiology of the complication.

In the cases of renal disease originally described by Bright[1] (1836) acute pericarditis is listed in 8 out of 100 autopsies. He regarded it as part of a general tendency toward involvement of the serous membranes. In the same series the pleura was acutely involved in 16 cases and the peritoneum in 12. Special emphasis upon the frequency of pericarditis in chronic nephritis was given by Taylor[2] (1845), who described it in 5 out of 51 cases that he examined post mortem. It is interesting to note in passing that five years later Taylor concluded that whereas practically all the cases of rheumatic pericarditis got well those following renal disease were uniformly fatal. Von Bamberger[3] states, according to his own observations, that 14 per cent of cases of Bright's disease develop pericarditis. The incidence given by other writers varies. Rosenstein[4] 7 per cent, Frerich[5] 4.5 per cent, M. Rayer[6] 5.4 per cent. Sibson[7] made an exhaustive study of the subject and in 1691 collected cases of nephritis found the incidence 8.1 per cent. In his personal series of 285 cases there were 25 cases of acute pericarditis, or 8.8 per cent. Sibson mentions 2 cases of this latter group occurring in acute nephritis and the remainder in chronic nephritis.

In the autopsy group here presented of 162 cases of nephritis 18 had acute pericarditis, or 11.1 per cent. In a clinical group represented by cases of nephritis admitted to the hospital during the corresponding period, out of 929 cases there were 30 who developed pericarditis, or 3.2 per cent. The latter figure may be said to express the incidence of the complication in non-fatal and fatal nephritis, the autopsy statistics naturally representing the incidence in fatal cases alone. Approached from the viewpoint of the causes of pericarditis in general, Sears[8] found chronic nephritis operating as a factor in 7 out of 100 cases, or 7 per cent; Preble,[9] analyzing 300 cases in 11.2 per cent. Locke[10] studied pericarditis as it occurred in 3683 routine autopsies; 4 per cent of all autopsies showed an acute pericarditis. Evidence of this complication in the clinical notes was present in 17 per cent. In 150 autopsies of acute pericarditis 16 were associated with nephritis, or 11 per cent. Of these 8 were grouped as fibrinous pericarditis, 6 adhesive (total or partial) and 2 purulent. Hirschfelder[11] states that pericarditis occurred in 230 (1 per cent) of the cases admitted to the medical service of the Johns Hopkins Hospital. Of these 33 were associated with nephritis, or 14.3 per cent. According to the statistics of these authors,

nephritis comes third as a cause of pericarditis, pneumonia and rheumatism only being more frequent. Sinnhuber[12] places rheumatism first, nephritis second, and the pyogenic infections afterward.

Concerning the pathogenesis of this complication in nephritis standard text-books are either non-committal or at variance with each other. Whether the cause lies in chemical irritation or secondary infection remains undetermined, as expressed by Gibson.[13] Aschoff[14] makes a similar statement. MacCallum,[15] speaking of the frequent occurrence of pericarditis in nephritis, says, "In these cases it is generally difficult to find any bacteria." Hirschfelder[16] states that "it is usually due to an intercurrent infection, and the pyogenic cocci can often be cultivated from the exudate." According to Wells[17] "the uremic pericarditis and endocarditis, which have often failed by ordinary methods to yield any bacteria, are apparently toxic processes." Sinnhuber[12] (1911) says that since neither cultures nor microscopic researches have shown bacteria it is apparently a toxic inflammation.

The clinical group comprises 30 cases. As all the factors studied were not present in every case the proportion of each will be mentioned. The laboratory data collected were selected at or near the onset of the pericarditis in order that they should represent factors which perhaps predisposed toward the complication. They are not then the most extreme findings, and are not at all in most instances antemortem statistics. This will be seen more clearly when the duration of life, after development of the pericarditis, is discussed.

The patients were mostly just below middle age, the average age being thirty-seven years, the extremes fifteen years and fifty-seven years. There were an equal number of males and females. All were diagnosed chronic nephritis. No case of acute nephritis developed the complication, nor was there a case of pure salt-and-water retention nephritis (nephrosis). The cardinal features included a uniformly marked elevation in blood-pressure; a severe secondary anemia; moderate leukocytosis and polynucleosis; a distinct hemorrhagic tendency; a greatly increased blood-urea nitrogen; minimal phenolsulphonephthalein excretion and a conspicuous acidosis.*

* Acidosis is used here to mean a lowering of the bicarbonate reserve of the blood and does not necessarily indicate any change in the H-ion concentration. In these cases the diminution in available alkali no doubt arose through an increase in non-volatile acid. It is, of course, impossible to say by a determination of the plasma CO_2 whether the H-ion concentration of the blood persisted normal or underwent an actual shift in the acid direction, *i. e.*, whether the acidosis was compensated or uncompensated. (Terms originally used by Hasselbach, K.A., and Gammeltoft, S.A., Biochem. z. 1915, 68, 205, and adopted by Van Slyke, Donald D., and Cullen, Glean E., Jour. Biol. Chem., 1917, 21, 293). This important point can in the future be satisfactorily determined by constructing the so-called carbon dioxide diagram of the blood. (Haggard and Y. Henderson (Jour. Biol. Chem., 1919, 39, 163). It has recently been applied to a series of hospital patients by Means, Beck and Woodwell (Jour. Exp Med., 1921, 32, 201), and its value in determining actual changes in blood reaction has been demonstrated.

The average blood-pressure of the 30 cases was 214/136, the lowest 170/110. The average red blood-cell count of 22 tested cases was 2,738,000, the hemoglobin 48 per cent. In no case was there absence of anemia. The majority of the cases showed a moderate leukocytosis, with polynuclear predominance at the time of development of the pericarditis. The average of 29 cases was 15,900 for the white count, polynuclears 87 per cent; 2 cases only showed a white blood count under 10,000. The temperature was variable, often showing a slight elevation, rarely more than 1° or 2°. A distinct tendency to hemorrhage was noted in 21 of the 30 cases, or 70 per cent. This varied in degree and was manifested in various ways, commonly as petechiæ, purpura of the skin or mucous membrane, epistaxis, hematemesis and rectal hemorrhages.

The average blood urea of 28 cases was 2.69 gm. per L. All were considerably elevated except one case, (No. 29), which had 0.45gm per L. This patient had general arteriosclerosis with a blood-pressure of 300/200, albumin and casts in the urine without fixation of the specific gravity, and, in addition, aortic valvular heart disease. The pericardial friction lasted four days and the patient improved and left the hospital. He died one and a half months later at home. The nephropathy here would perhaps be grouped as an arteriosclerotic kidney. Elevation of the blood urea in cases of nephritis with acute pericarditis has been commented upon by French authors (in the French, "pericardite brightique"). In a series of 11 cases of Widal and Weil[18] the average was 2.71 gm. per L. Chalier and Novi-Jusserand[19] report 1 case in full in which the blood urea was 2.10 gm. per L. Other individual cases are mentioned by d'Ulrici,[20] 4.85 gm. per L., Froment and Rochaix,[21] 3.57 gm. per L., and Foy[22] 2.54 gm. per L. In comparing the average of this series, 2.69 gm. per L., it must be remembered that this value does not represent the maximal values of the cases, but the finding on or near the day when the pericardial rub first became evident. As will be emphasized later the blood urea progressively mounted. (See Table I for clinical data.)

In all of the 16 cases in which observations were made an acidosis, as evinced by a lowering of the plasma bicarbonate, was present. In 9 cases the figures were obtained by the carbon dioxide combining power of the plasma by the method of Van Slyke,[23] and averaged 28.7 vol. per cent. In 7 earlier cases the results are recorded by the carbon-dioxide tension of the alveolar air (Fridericia method), and averaged 24.59 mm. Hg. The lowest observation was 11.8 vol. per cent, plasma CO_2. (Some of the cases at times showed lower figures than are listed in the table, but here too those were selected which had the nearest time relationship to the onset of the pericardial friction.) A conspicuous diminution of the available blood alkali is thus evident from both methods. That most patients with advanced nephritis, especially in the last stages of

TABLE I.—CLINICAL DATA.

Case.	Age.	Sex.	B. P.	W. B. C.	Diff. polys., per cent.	R. B. C.	Hgb., per cent.	Bl. urea gms. per L.	Bl. CO_2	Phthalein, per cent.
1 E. P.	48	F	214/105	10,000	88	2,200,000	60	3.92	31.2 vol. per cent	
2 W. D.	50	M	216/150	13,000	88	1,000,000	15	2.50	26.6 mm. Hg.	8.0
3 J. G.	36	M	224/130	24,000	92	3,000,000	47	2.50	19.7 "	0
4 C. L.	49	F	200/140	18,200	92	3,200,000	64	3.73	.....	0
5 D. V.	44	M	208/120	10,000	..	3,000,000	50	2.70	10.2 "	0
6 W. F.	26	M	226/140	19,000	86	2,620,000	52	2.91	.....	0
7 C. A.	20	F	215/145	29,000	89	3,350,000	50	3.07		
8 M. E.	40	F	214/130	.....	..	.....	..	1.50		
9 T. L.	44	M	222/150	15,200	88	3,730,000	70	3.87	38.5 vol.	0
10 C. K.	15	M	200/130	23,700	80	2,912,000	40	4.45	22.1 "	0
11 A. K.	40	F	205/130	0,000	75	3,480,000	70	3.11	24.0 "	0
12 W. S.	37	M	170/135	14,000	80	4,000,000	55	1.58	47.0 "	15
13 E. S.	21	F	170/110	11,200	80	1,700,000	20	4.08	32.8 "	
14 T. S.	50	M	220/122	13,000	80	3,500,000	50	4.12	21.4 "	1
15 L. M.	19	F	180/110	10,200	68	1,300,000	30	2.05	11.8 "	
16 T. S.	48	M	218/110	23,800	91	3,400,000	55	1.85	25.2 mm. Hg.	7
17 H. W.	19	F	178/110	11,500	88	1,475,000	32	2.50	22.75 "	
18 A. W.	40	F	244/152	13,800	84	.....	..	2.61		
19 R. A.	48	M	210/148	12,000	67	.....	..	1.77	.....	34
20 M. G.	36	F	220/180	9,400	82	.....	50	3.72	.....	0
21 L. L.	38	M	210/138	14,100	72	.....	70	2.20	.....	10
22 H. P.	44	M	210/140	20,000	83	.....	..	....	.....	10
23 L. J.	36	F	170/90	8,460	74	1,480,000	30	....	.....	3
24 R. T.	35	F	195/100	17,000	90	1,828,000	38	2.44	30.5 vol. per cent	0
25 E. S.	32	F	255/175	19,800	92	2,380,000	45	3.14		
26 B. G.	37	M	230/150	15,150	89	4,600,000	69	1.60		
27 E. H.	46	F	250/140	14,500	74	3,500,000	46	1.75	.....	6
28 J. E.	57	M	212/140	17,200	90	.....	..	1.14	29.45 mm. Hg.	9
29 L. H.	42	F	300/200	15,000	76	.....	..	0.45	.....	15
30 V. F.	20	M	228/162	19,000	95	2,584,000	42	2.27	29.25 "	
Average	37.8	M50%	214/136	15,036	87	2,738,409	48	2.69	28.7 vol per cent 24.59 mm. Hg.	5.0

the disease, have a demonstrable acidosis has been shown by Henderson and Palmer,[24] Peabody,[25] Chace and Myers[26] and others. According to the figures of this series 100 per cent of the tested cases had an acidosis at the time of onset of the pericarditis, in most instances of severe grade, represented as it was by an average plasma CO_2 of 28.7 vol. per cent.

The average phenolsulphonepthalein excretion in 20 cases was 5.9 per cent; in one-half of these it was zero. No proportional relationship could be established between the degree of acidosis, the elevation in blood urea or the excretion of phthalein. This is in accordance with the similar earlier observation of Peabody.[25]

In 26 of the 30 cases a pericardial friction-rub was described in the clinical notes. In 1 additional case the diagnosis was made by signs of effusion. The diagnosis was thus made clinically in 90 per cent of the cases. The frequency with which the diagnosis was made by a friction-rub may perhaps be accounted for by the small amount of pericardial fluid that is present in this type of pericarditis (as will be seen later). The average duration of the rub was eight days;* in one case it could be heard for two and a half months. The average length of life after the appearance of the rub was twenty-nine days. One patient, in whom the pericardial friction lasted nine days, lived for one year thereafter. If this case were not included the average length of life after the rub became evident would be sixteen days. Another patient lived two months after the onset of the pericardlitis; a third two and a half months, and a fourth four months. This is mentioned to emphasize that the pericarditis is not always a terminal complication. It may subside and the patient undergo a remission of symptoms, leave the hospital and perhaps live comfortably for several months. The case mentioned above that lived for one year showed at autopsy an adherent pericardium.

The pericarditis does not seem to be the direct cause of death in many of these cases. In some of them it seems related rather to the increasing retention of nitrogen in the blood. The acidosis too does not appear responsible for the final succumbing of the patient. In these cases it was generally combated with sodium bicarbonate whenever it became dangerously low. The case of L.M. may be taken as an example: At the time of her admission to the hospital, when the pericarditis was first noted, the blood urea was 2.95 gm. per L. and the plasma CO_2 11.8 vol. per cent. The friction-rub persisted until death, sixteen days later. Bicarbonate was persistently administered, aiming to keep the blood CO_2 at least above

* It must be remembered that the duration of the rub applies to hospital residence. In some cases the rub was present on admission and an accurate estimation of its actual duration is thus not possible.

40 vol. per cent. The determinations ran as follows: 11.8 vol. per cent, 10.9 vol. per cent, 27.1 vol. per cent, 48.1 vol. per cent, 40.4 vol. per cent, 46 vol. per cent. At the same time the blood urea mounted 2.95 gm. per L., 3.17 gm. per L., 3.55 gm. per L., 4.75 gm. per L. The cause of death did not seem linked with progressive heart failure nor obviously with the acidosis, but rather with the progressive retention of nitrogen in the blood.

Of the 30 cases 18 came to autopsy. The kidney lesion was predominantly the contracted granular kidney, more commonly the glomerular than the arteriosclerotic contracted kidney. Sibson's[7] cases occurred very largely in the small granular kidney. Lecorche and Talamon[27] also believed pericarditis to be more frequent in this interstitial form of the disease. According to Grainger Stewart[28] however, it was equally common in the parenchymatous and renal cirrhosis. No attempt has been made in this series to correlate the anatomical lesions with the clinical grouping of the nephritis.

The pathology of the pericarditis possesses special interest because of its relation to some of the bacteriological findings. The pericardial cavity contained in most instances a little increase in fluid, 12 of the 18 cases having between 50 and 200 cc; 2 showed no fluid; 1 had an adherent pericardium; 3 had a large excess of fluid; in 1, 700 cc of bloody fluid; another that had been tapped during life contained 2000 cc of dark red fluid in the pericardial cavity. Chalier and Novi-Jusserand's[29] case had 250 gm. and Widal and Weil's[30] cases between 50 and 250 gm. of pericardial fluid. The fluid was usually of a light or dark amber color, clear or only slightly turbid. Fibrinous adhesions were almost always present, appearing either as thin delicate bands or as a fine fibrinous deposit. Microscopically the fibrin network was free from cells beyond occasional scattered round cells. The epicardium, as a rule, was a little thickened and in the majority of cases contained a slight to a moderate infiltration of small mononuclear cells—in a few instances no infiltration at all. In 2 cases the mononuclear infiltration extended into the muscle substance. In 5 cases, however, a polynuclear infiltration was present, with smaller numbers of mononuclear cells as well. In 4 of these the polynuclear infiltration was present in the pericardium. In 1 case the pericardium was infiltrated moderately with mononuclear cells, but the smear of the pericardial exudate showed pus, blood cells and bacteria. Of the 5 cases showing polynuclear infiltration 4 were cultured, and all of these showed the presence of pyogenic organisms. The fifth had a sterile blood culture, but no culture had been made from the pericardium. In 2 of the cases the organisms were recovered in pure culture, one a hemolytic Staphylococcus aureus and the other a short-chain streptococcus. The third showed Staphylococcus albus and B. coli communis; the fourth, a non-hemolytic streptococcus, a bacillus belonging to the

Friedländer group and a Gram-negative bacillus which did not form gas with dextrose. In 2 of the cases in which a blood culture was done at the same time the same organisms were recovered, indicating there was a terminal septicemia, the pericardium constituting a localization of the infectious process. In the remaining 12 cases in which polynuclear infiltration was not demonstrated there were 4 cases that were tested bacteriologically and the pericardium found free from infection in all. There was 1 of these cases that had a terminal bronchopneumonia in which the lung culture at autopsy yielded a pneumococcus IV, but in which the pericardium was sterile both in aërobic and anaërobic culture. (See Table II for bacteriological and pathological data.)

It is interesting even in this small group that the 4 cases with frank infection showed a polynuclear response in the epicardium or in the pericardial exudate, whereas the 4 cases in which no infection of the pericardium was present showed an absence of polynuclear leukocytes, and, instead, mononuclear infiltration or no cellular infiltration whatsoever. Of the 4 sterile cases, in 3 the cellular response in the pericardium varied from just a few scattered mononuclear cells to a moderate infiltration. In the fourth only the pericardial fluid and fibrin tags were available, and these showed no cells at all. This division of cases brings forth the obvious suggestion that there may actually be two distinct etiologies for the complication, an infectious and a non-infectious. Before this is further discussed the bacteriological findings in the literature as well as the experimental evidence will be reviewed. This has been delayed up to this point in order that the histology of the pericarditis might be compared with the bacteriological findings on the basis of the above results. No purposeful comparison has previously been made, nor do many of the reports contain the material for doing so.

The review of all authentic reports concerning the bacteriology of pericarditis in nephritis shows 33 in which the bacteriology of the pericardium was investigated (31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42). Except for the more recent article by Widal and Weil most of the studies were made before many of the more recent methods of bacteriological research were instituted, and therefore have only a limited value. Of these cases, however, 18 were sterile and 15 showed bacteria, usually of the pyogenic variety. The most frequent invader was the pneumococcus and the next frequently found was the Bacillus coli and the streptococcus. The attempt to link up the histology of the pericardium with the bacteriological findings in the cases previously reported yields a partial confirmation of the results in our series. When infection was present polynuclear leukocytes were described in the pericardium or pericardial exudate in each instance in which individual case reports were given.

TABLE II.—PATHOLOGICAL AND BACTERIOLOGICAL DATA.

Case.	Duration of rub, days.	Length of life after onset of pericarditis days.	Chest involvement.	Tendency to hemorrhage.	Pathology of pericardium.	Bacteriology.
1 E. P.	1	3	Noae	Moderate hematemesis blood in stool.	Autopsy by Dr. Lamb, 100 cc pericardiol fluid, amber, ond slightly turbid. Straads of fibrin cover pericordial surfaces. Petechini hemorrhages on visceral pericordium. Microscopicol lymphocytes, poly infiltration ond edema.	Smeors from pericardial fluid at autopsy showed a non-hymolytic streptococcus, a bacillus belooging to the Friedländer group, and a gram negative boeillus which does not form gas with dextrose. (Air wns found in blood vessels in this ease.)
2 W. D.	3	3	Pleurisy	Morked; rectal hemorrhoge; brain hemorrhage; skin petechiæ.	Autopsy by Dr. Evans. Large exeess of cloudy yellow fluid. Fine diffuse fibrinous deposit. Microscopical fibrin is scanty and contains o few round eells.	No cultures of pericardium.
3 J. G.	4	4	Hydrothorox	Morked; rectal hemorrhoge. Epistaxis	No autopsy.	Two blood cultures during life sterile. No cultures of pericordium.
4 C. L.	4	4	None.	Moderate; hemorrhage in iotestioes nod stomoch; petechiæ on skin.	Autopsy by Dr. Meierhof. Fine fibriaous exudate over port of pericardium. Microscopical o very few eells ore seen, of small mononucleor type.	No cultures of pericardium.
5 D. V.	8	8	Pleurisy.	Moderate; rectal hemorrhage; petechia on skin.	Autopsy by Dr. Mueller. Fresh fibriaous exudote. About 200 ee pericordial fluid. Abundont infiltration of small ond lorge monoaueleor eells, wbich extend into muscle. Few scottered polys.	No cultures of pericardium.
6 W. F.	(1)	..	Pleurisy.	Moderate: bleeding gums; epistaxis.	Autopsy by Dr. Mackenzie. 150 to 200 ee rather dork. slightly turbid, nmber fluid. Fresh grouulnr fibrin. Microscopical numerous smoll mononucleor eells. Occasional poly.	No cultures of pericardium.
7 C. A.	1	1	Hydrothorax.	Very slight.	No autopsy.	

[1] Diognosis not made clinicolly.

8	M. E.	3	3	None.	None.	No autopsy.	Blood culture showed pneumococcus II. Pleural fluid also pneumococcus II. No cultures of pericardium.
9	T. L.	4	8	None.	Marked; rectal hemorrhage; hemnturia; epistaxis.	Autopsy by Dr. Stier. No fluid. Fine fibrinous adhesions. Muscle shows scattered areas of whitish color and smaller areas of darker red color, apparently hemorrhages. Microscopical fibrinous ndhesions contain very few cells. Beneath them nre numerous small mononuclear cells, with an occasional poly.	By Miss Cooper. Lung culture nt autopsy sterile. No pericnrdial culture.
10	C. K.	13	13	Pleurisy.	Marked; epistaxis; hemorrhago from bladder and into skin.	Autopsy by Dr. Eggstein. Increased pericnrdial fluid. Firm white fibrinous masses in some places in others deliente fibrinous exudate. Microscopical loose fibrinous network, with only nn occasional scattered small mononuclear.	No cultures.
11	A. K.	1	1	Hydrothornx.	Slight; bleeding gums.	No nutopsy.	No cultures.
12	W. S.	9	1 yr.[2]	Pleurisy.	None.	Autopsy by Dr. Eggstein. Pericardium is adherent, and shows considerable mononuclear infiltrntion.	No cultures.
13	E. S.	5	5	None.	Very mnrked; bleeding from gums, stomach, rectum, and vaginn; epistaxis.	No nutopsy.	No cultures.
14	T. S.	3	5	None.	None.	No nutopsy.	No cultures.
15	L. M.	16	16	None.	Marked; hematemcsis: epistaxis.	Periencrdial puncture. Clear amber fluid with fibrin shreds. No cells in smenr; no autopsy.	Pericardial fluid aspirated three hours after death sterile.
16	T. S.	(3)	..	Hydrothorax.	None.	Autopsy by Dr. Meierhof. 100 cc of yellow turbid fluid. Fresh fibrinous ndhesions between parietal and visceral pericardium. Microscopical fibrin tngs nro without cellular infiltrntion. Lymphocytes present beneath epicardium.	By Miss Frazier. Culture of lung at autopsy showed a pneumococcus II. Culture of pericardium was sterile, aërobically and anaërobically.

[2] Died one year later. Autopsy showed an adherent pericardium.

[3] Diagnosis not made clinically.

TABLE II.—PATHOLOGICAL AND BACTERIOLOGICAL DATA.—(*Continued*)

Case.	Duration of rub. dnys.	Length of life after onset of peri-carditis days.	Chest involvement.	Tendency to hemorrhage.	Pathology of pericardium.	Bacteriology.
17 H. W.	(*)	14	Pleurisy hydrotborax.	Moderate; epistaxis.	Autopsy by Dr. Evans. Pericardial fluid, dork red, 2000 cc. Fine fibrinous deposit. Petechial hemorrhages over tbe pericardium. Microscopical small round cells present beneath the fibrin. (Tapped during life.).	By Miss Olmstead. Blood culture during life sterile. Blood culture ot autopsy sterile. Smear or pericardial fluid obtained by tapping during life showed no organisms.
18 A. W.	7	7	None.	Moderate; petechiæ; hemorrbages io conjuoctiva; blood in colon irrigation.	Autopsy by Dr. Eggstein. Pericardium covered by maoy shaggy irregular fibrinous adhesions. These are bright red with a few areas of groyish mottling suggesting pus. In places fibrin 1 cm. thick. Microscopical exudate consists of polys, fibrin, red cells and masses of bacteria.	Autopsy culture of spleen and liver showed hemolytic staphylococcus aureus. Smear of heart showed many Gram-positive coeci, probably staphylococci.
19 R. A.	1	1	Pleurisy	None.	Autopsy by Dr. Mueller. Light fresh fibrinous exudate. Slight increase in fluid which is rnther turbid. Fibrous layer of epicardium the seat of a moderote infiltration of polyouclear leukocytes and o few round cells.	By Miss Olmstead. Pericardial culture showed B. coli communis and staphylococcus albus. Gram-negative bacilli aod Grain-positive coccus were seen on the smear.
20 M. G.	3	3	None.	Moderate; epistaxis; splittiog up of blood.	Autopsy by Dr. Evans. Fine fibrinous exudate. No fluid. Epicardium distorted. It is edematous, infiltrated with plasmo cells, polys, small round cells; aod shows o fibrinous deposit.	By Miss Olmstead. Blood culture at autopsy sterile. No culture of pericardium.
21 L. L.	8	8	Pleurisy; hydrotborax.	Shown ot outopsy; hemorrbages in brain, stomach nnd bladder.	Autopsy by Dr. Stillmoo. Brown turbid fluid, oot io excess. Many flakes of fibrin over o dull pericardium. No cellulor infiltration.	No pericordial culture.

* Diagnosed by signs of effusion, fourteen days before death.

22	H. P.	4	4	Pleurisy.	None.	Autopsy by Dr. Pappenheimer. 100 cc pale yellow fluid. Large flakes and shreds of fibrin. Fresh adhesions; microscopical epicardium not seen.	No pericardial culture.
23	L. J.	80	80	Pleurisy.	Moderate; epistaxis; coughed up blood.	Autopsy by Dr. MacCallum. 700 cc bloody fluid. Thickened pericardium. Soft fresh fibrinous exudate. Pericardium is infiltrated moderately with mononuclear wandering cells and infrequent polys. Fibrin almost free from cells. Smear of pericardial fluid: pus, blood cells and Gram-positive cocci.	By Dr. Soper. Smear of pericardial fluid showed pus, blood-cells, and Gram-positive short-chained cocci. Culture was pure streptococcus.
24	R. T.	8[6]	29	No autopsy; pleurisy.	Moderate; hematemesis; epistaxis.	No autopsy.	No culture of pericardium.
25	E. S.	2	2	None.	None.	No autopsy.	No culture of pericardium.
20	B. G.	3	3	None.	None.	No autopsy.	No culture of pericardium.
27	E. H.	(8)	..	Pleurisy; hydrothorax.	Slight; vomited clots of blood.	Autopsy by Dr. Evans. No excess of fluid. Delicate fibrinous deposit. Small mononuclears in epicardium.	No pericardial culture.
28	J. E.	20	120[7]	Hydrothorax.	Slight; rectal hemorrhage.	No autopsy. (Patient went home improved. Died three months later.)	No pericardial culture.
29	L. H.	4	60	None.	None.	No autopsy.	No pericardial culture.
30	V. F.	2	2	None.	Marked; purpura of skin and mucus membranes; bloody urine.	Autopsy by Dr. Allison. Thin fibrinous adhesions; 50 to 100 cc clear fluid. Leukocytic infiltration, mostly mononuclears, extending a short way into muscle.	By Miss Olmstead. Pericardial culture sterile.
Average		8	29	56.6 per cent.			

[6] Rub lasted eight days. Patient then left the hospital. Died three weeks later.

[7] Rub lasted twenty days and then disappeared. Patient left the hospital improved. Died three months after departure, four months after disappearance of rub.

[8] Diagnosis not made clinically.

The experimental production of acute pericarditis in animals by simulating the kidney damage in man has been productive largely of negative results. The ligature of both ureters or double nephrectomy is not followed by pericardial inflammation (Banti,[43] Keraval,[44] Beco,[45] Chatin and Guinard[46]). (One out of the five rabbits in Beco's series developed an acute serofibrinous pericarditis following ureter ligature. The pericardial fluid was sterile, but Beco admits the case is a bizarre one and does not prove the chemical theory.) Banti[47] also performed these experiments by first producing a point of lowered resistance through cauterization of the pericardial surface. An acute pericarditis develops which is more marked and of quicker onset than the control pericarditis that follows cauterization alone. This evidence, however, can hardly be accepted as proving anything definite. Keraval[48] injected urea and ammonium carbonate into the pericardial cavity without causing pericarditis. Chatin[49] found the serum of cases of "pericardite brightique" hypotoxic to animals rather than hypertoxic. These authors, because of the negative clinical bacteriology, nevertheless believed chemical irritation to be the main cause of pericarditis in chronic renal disease.

The observation of Flexner[50] upon terminal infections occurring in chronic diseases such as cirrhosis of the liver, diabetes mellitus and chronic nephritis shows that a local or general invasion by bacteria is frequently the immediate cause of death. Pericarditis, however, is especially characteristic as a complication of nephritis and not of other chronic affections. Although it may occur occasionally as a terminal infection in any chronic disease, there is an additional frequency in renal disease unexplained by the factors of infection.

Further evidence upon the etiology of this complication is of indirect nature. To determine whether the pericarditis had any relation to involvement of the other serous membranes, the incidence of involvement of the pleural cavity was tabulated in the cases that had pericarditis. Fibrinous pleurisy was present in 25 per cent, fibrinous pleurisy or hydrothorax in 56.6 per cent, peritonitis in none.

In the interest of the chemical theory an analogy may be drawn from the reactive pericarditis that follows infarction of heart muscle as after coronary closure. Here presumably a sterile chemical irritant derived from the products of muscle necrosis produces the pericarditis. Fischer[51] (1897) in fact explained the nephritic pericarditis upon very similar reasoning. He proposed that a myocarditis existed in uremia and that the irritant effect of the damaged muscle cells caused an inflammation in the adjoining serous layer of the pericardium. Rhomberg[52] has found in simple endocarditis thrombosis of the myocardial vessels and myocardial degeneration. Stengel[53] would couple this with what occurs in pericarditis, believing

that the symptoms of the latter are due to the underlying myocardial disease. Changes in the muscle fibers were observed in many of the cases of this series and occasionally mononuclear leukocytes were seen to invade the muscle nearby; but there was not enough of constancy either in location or character of the pathology to warrant any conclusions being drawn.

Comment. There are, then, two distinct theories that have been advanced to explain the etiology of this complication: (1) bacterial and (2) chemical. On the basis of the bacterial origin it may be stated that in a small group of cases microörganisms have been cultured from the inflamed pericardium. These organisms are of the same nature as those found by Flexner in the general and local infections of chronic renal disease. Furthermore the characteristic response to infection is shown by the presence of polynuclear leukocytes in the pericardium or pericardial fluid. This histology of the infected cases is demonstrated in the present series and in the infected cases reviewed in the literature. The evidence seems definite that in a small proportion of cases infection is the cause of the pericarditis.

In support of the chemical origin of the complication, the greatest argument is perhaps the existence of cases that have been found sterile after careful culture. Not only have aërobic and anaërobic cultures been made on very diverse media, but the pericardial fluid has been injected into animals, subcutaneously and intraperitoneally, with negative results. An additional important consideration is the non-infectious nature of the toxemia. The presence of a marked elevation in blood urea has been commented upon by French observers. Our results confirm the nitrogen retention and show in addition that there is simultaneously a notable acidosis and that both findings are present at the onset of the pericarditis. The secondary anemia was constantly present and generally severe. The tendency to hemorrhage was a conspicuous feature. As further evidence the differing pathological histology of the sterile group may be stressed. The pericardium of the sterile cases showed no infiltration with polynuclear leukocytes, but instead the presence of mononuclear leukocytes, or, infrequently, no cellular infiltration whatever. The remainder of the cases not cultured showed the pathological characteristics of the sterile group. This histology of the sterile group finds partial confirmation in the literature. In Chatin's carefully described case, mononuclear infiltration in the pericardium is fully noted; not so the cases from Banti's clinic, in which, however, the histology is incompletely given. The presence of round cells in an inflammatory area brings forth the possibility of a tuberculous etiology. Tuberculosis of the pericardium is not uncommon. However, this was not the pathology here. The mononuclear leukocytes were more likely to be scattered than localized; there were no tubercles, giant cells or characteristic granulation

tissue. The suggestion of a chemical irritant as cause of the majority of the cases is thus entirely compatible with the chemical nature of the toxemia and with the bacteriology and pathology of the condition. A clear-cut example of a pericarditis of non-infectious and therefore chemical origin is found in the acute pericarditis that follows infarction of heart muscle.

The possibility that both factors may have been present in an individual case presents itself. A pericardium previously inflamed, in response to a chemical agency, may finally become the seat of a terminal infection. For this reason sharp division of the cases may not always be possible. Case No. 23 may be mentioned as perhaps an example and the facts briefly outlined. In this case the pericardium showed a mononuclear infiltration, whereas the pericardial fluid contained pus and numerous cocci. The culture gave a pure streptococcus representing apparently a terminal infection. And yet the patient had had pericarditis for two and a half months before death. None of the other infected cases lived longer than seven days and the average was less than four days. Of the proved sterile cases, three were diagnosed and the average length of life after the onset of pericarditis was over ten days. The remainder of the cases fell pathologically into the sterile group and gave a considerably longer duration of life. The paucity of the proved cases and the complexity of the other findings limit the value of further comparisons.

Summary. 1. A description is given of the clinical and laboratory characteristics of a group of 30 cases of chronic nephritis at the time of development of an acute pericarditis. A marked nitrogen retention in the blood, a constantly present acidosis, a high blood-pressure, severe secondary anemia and a tendency to hemorrhage were conspicuous features.

2. It is pointed out that pericarditis is not a terminal complication in the sense that it always terminates the life of the patient. The average duration of life after the onset of the pericarditis was twenty-nine days. Excluding a patient that lived one year thereafter the average figure was sixteen days.

3. Death in many of these cases did not seem linked with an advancing heart failure or to the acidosis, but rather to the progressive retention of nitrogen in the blood.

4. Except in one case the diagnosis was made by the presence of pericardial friction and not by the signs of effusion. The diagnosis was made clinically in 90 per cent of the cases.

5. In 4 cases direct culture of the pericardium yielded pyogenic organisms. In 4 other cases culture of the pericardium was sterile. In the infected cases the cellular infiltration of the pericardium was predominantly of the polynuclear type; in the sterile cases the infiltration was predominantly of mononuclear type. The cases in which the pericardium was not cultured showed the pathological characteristics of the sterile group.

6. Reasons are given for believing that the majority of cases of pericarditis in chronic nephritis are of non-infectious origin. The assumption of a chemical irritant as cause of the complication is entirely compatible with the chemical toxemia of the patient and with the bacteriological and pathological findings in the pericardium.

7. A small group of cases exists in which frank infection of the pericardium is present. It is believed that the pericarditis in this group is generally of true infectious etiology. The possibility is present, however, that the complication may sometimes be of chemical origin and the inflamed pericardium become secondarily infected.

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BILIARY TRACT DISEASE: SOME LESSONS LEARNED FROM DUODENOBILIARY DRAINAGE. FUTURE PROBLEMS. CITATION OF CASES.*

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Four years have now elapsed since the late Professor Samuel J. Meltzer announced the effect that magnesium sulphate produced on the duodenum and on Oddi's sphincter of the common duct when locally introduced,[1] and four years have likewise elapsed since this

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